EDITORIAL

The right atrium as an anatomic set-up for re-entry: electrophysiology goes back to anatomy

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Rapid, complete, uniform activation of all atrial tissue is important for rhythm stability. Typical flutter, the most common among atrial tachycardias, has anisotropic conduction at the terminal crest as an essential part of its mechanism

Normal heart activation is characterised by regular alternation between depolarisation/repolarisation and rest. Activation originates in the right atrium, around the sinus node area, and spreads until the atria are completely activated. Once activation is complete atrial tissue is refractory and a period of rest necessarily precedes the next activation cycle. This sequence is caused by pacemaker cells with discharge rates that respond to neural and humoral stimuli, thus allowing adaptation of heart function to physiologic demands. However, this normal sequence is not so simple to sustain, as demonstrated by the frequent appearance of re-entrant tachycardias.

Rapid, complete, uniform activation of all atrial tissue is important for rhythm stability. Preferential conduction pathways have been long recognised in the atria,[1–3] despite the absence of bundles of specialised conduction akin to the His-Purkinje network of the ventricles.[4] James and Sherf[5] attributed the faster conduction along the terminal crest and Bachmann's bundle to the presence of specialised cells; however, present day thinking explains this by the anisotropic properties of atrial myocardium.

ANISOTROPY

In cardiac electrophysiology "anisotropy" refers to changes in conduction dependent on the anatomic orientation of myocardial fibres. According to the cable theory, conduction velocity depends on myocardial action potential upstroke—that is, the velocity of intracellular voltage change from negative to positive at the time of depolarisation.[6] A decrease in the rate of depolarisation would cause conduction slowing as, for example, when antiarrhythmic drugs block the fast sodium (Na^+) channel. But a new approach was developed in the early 1980s when Spach showed that in atrial myocardial bundles packed longitudinally (pectinate muscle fragments) conduction could be faster along the main axis of the bundle than across it, despite a slower rate of action potential upstroke.[7] The importance of intercellular coupling in determining conduction then began to be understood. Low resistance intercellular end-to-end connections are responsible for faster conduction along the longitudinal axis of the fibres, with respect to conduction across this axis. This effect can be most significant in complex structures where fibres change directions at sharp angles or cross in different directions.[8] The effect of fibre "packing" is enhanced by local differences in gap junction distribution. Saffitz and colleagues[9] showed that in ventricular myocardium, gap junction distribution is fairly even end-to-end and side-to-side. In this setting anisotropy is relatively modest, longitudinal conduction being three times as fast as transverse conduction. On the other hand, in the terminal crest gap junctions are almost exclusively disposed end-to-end and conduction velocity is 10 times faster in the longitudinal than in the transverse direction.

A normal, tightly packed, well organised atrial muscle structure can have an organising role for atrial activation, by helping rapid and uniform impulse conduction, thus preventing re-entrant activation. While Maze surgery directs activation linearly by creating barriers,[10] normal anisotropy could have the same effect by speeding up conduction along the preferred anatomic axis. But anisotropy also has its traps. Spach extended his observations to a number of specific anatomic singularities, such as bifurcations, crossings, and perpendicular bundle insertions, and he could show that anisotropy could account for slow conduction, unidirectional block, and re-entry.[7 8] And re-entry is the most frequent mechanism of tachycardias.

ATRIAL FLUTTER

Typical flutter, the most common among atrial tachycardias, has anisotropic conduction at the terminal crest as an essential part of its mechanism.[11–13] Re-entry is a simple concept, but not an easy one to picture in the real anatomy of the heart. A ring of tissue, such as Mines made by cutting the heart of a tortoise,[14] is ideal for continuous circular activation, because there is no way for activation to turn back on itself or cut across to the opposite side of the ring, disturbing the process. Although such a ring does not exist in the normal heart, a more-or-less circular band of myocardium can be formed by appropriately placed obstacles. In 1947 Rosenblueth and García-Ramos[15] created such a band in the right atrium of dogs by crushing the myocardium between the superior and inferior caval vein orifices (fig 1). In this preparation activation could rotate permanently between an anterior border, made by the tricuspid ring, and a posterior border made by the crushing injury and the openings of both caval veins.

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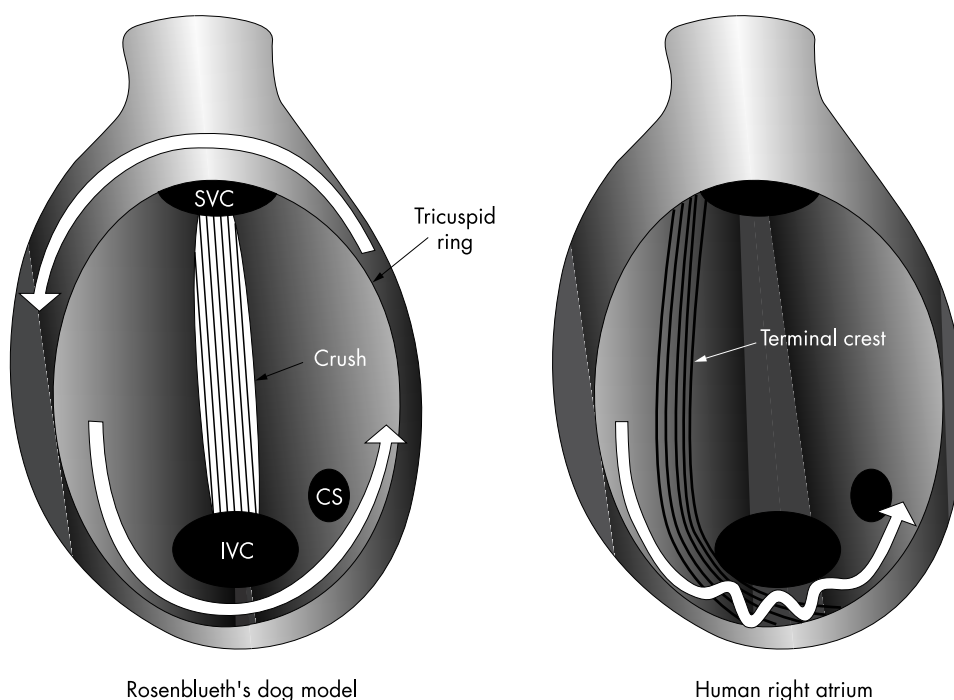Getafe, Madrid, Spain;
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Figure 1 Schematic representation of posterior right atrial wall barriers making macroreentry possible. On the left the crush injury made by Rosenblueth and García-Ramos[15] in their dog experimental model allows circular activation around the tricuspid ring. On the right the position of the human terminal crest is represented. Note that its longitudinal axis is a barrier to transverse conduction, akin to Rosemblueth's crush injury. Terminal branching of the crest at the inferior vena cava-tricuspid ring isthmus may also facilitate re-entry by slowing conduction locally (wavey arrow). IVC, inferior vena cava; SVC, superior vena cava.

When Puech described the typical flutter circuit[16] after his endocardial mapping data, he basically reproduced Rosemblueth's circuit, activation rotating around the tricuspid ring. However, no insight was offered on a posterior obstacle preventing short circuiting or turning back of activation. It is more recently that we and others[11 12] have been able to determine that in human typical flutter there is indeed a posterior obstacle, albeit a functional one, based on anisotropy at and around the terminal crest. Endocardial recordings obtained with catheters, precisely placed over the crest with echocardiographic guidance, reveal double potentials, reflecting separate activation on both sides of the functional line of block. Thus the terminal crest combines with the orifices of the superior vena cava (SVC) and inferior vena cava (IVC) to make the large posterior obstacle that binds the circuit posteriorly, just as with Rosemblueth's crushing injury (fig 1).

But the role of anisotropy in the flutter circuit may not end just here. Relatively slow conduction has been described in the *isthmus* between the anterior rim of the IVC and the inferior rim of the tricuspid ring (IVC-TV isthmus),[17] and this may be important both for initiation and maintenance of re-entry. Fine studies of fibre orientation in the isthmus[18 19] show terminal ramifications of the crest running perpendicular to insert in the tricuspid ring, thus making again a perfect anatomic set-up for local conduction slowing during circular activation (fig 1).

The study by Sánchez Quintana and colleagues[20] in this issue of *Heart* is a unique systematic description of the muscular and fibrous components of this structure in the human that will greatly help us understand the relations between anatomy and electrophysiology in this critical area. Particularly interesting is the complex relation between the tightly packed, parallel myocardium of the crest itself and the fibres making the pectinate muscles, connected to the crest in varying angles that should favour block during rapid activation. It has been proposed that in human flutter transverse block may occur close to, but not precisely at, the terminal crest level,[21] and recent experimental work showed how block can occur preferentially at the junction of the terminal crest with the pectinate muscles region.[22]

Sánchez Quintana and colleagues lament not having been able to study hearts which have borne atrial flutter throughout life. This is an important point, because with very few exceptions all human hearts have a tricuspid ring, an SVC, an IVC, and a terminal crest, but only very few develop flutter in their whole lives. Amazingly, very few data are available on the anatomy of the right atrium in flutter, including the presence and degree of dilatation and/or hypertrophy. Only preliminary intracardiac echocardiographic data[23] suggest that terminal crest thickness can be larger in patients with atrial flutter than in those with fibrillation, and this would fit nicely with the higher transverse blocking capability of the crest in patients with atrial flutter[24] compared with those with fibrillation. Fortunately atrial flutter is a relatively benign arrhythmia so that we will have to await a wide collaborative effort to obtain data on its anatomic bases, especially in relation with its more frequent relative, atrial fibrillation.

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IMAGES IN CARDIOLOGY..

Right coronary artery aneurysm diagnosed with multislice computed tomographic angiography

An 83 year old man presented with shortness of breath. Physical examination revealed a raised jugular venous pressure and atrial fibrillation with a rapid ventricular response. A provisional diagnosis of congestive cardiac failure was made and treatment with digoxin and diuretics initiated. A transthoracic echocardiogram demonstrated a large "doughnut" shaped extracardiac mass compressing and displacing the right atrium.

Non-invasive coronary angiography was performed using multislice helical ("spiral") computed tomography (CT). The breath hold required for a scan time of 31 seconds was well tolerated. Following contrast injection images were acquired using a gantry rotation time of 500 ms and retrospective electrocardiographic gating. Images were then reconstructed using the diastolic phase. Despite the lack of a regular R–R interval, excellent image resolution was obtained.

Axial transverse reconstruction (multiplanar reconstruction or MPR) CT coronary angiography (upper panel) demonstrated a right coronary artery aneurysm. The right coronary artery is shown arising from the aorta (Ao). The contrast filled lumen (An) of the aneurysmal portion of the artery is surrounded by unenhanced thrombus, which explained the echocardiographic appearances. The aneurysm is compressing the right atrium. A three dimensional reconstruction (using volume rendering techniques or VRT) shows the lumen of the aneurysm (An in lower panel) without the surrounding unenhanced mural thrombus. Although this type of "visual" reconstruction provides excellent information for surgeons, the treatment in this case was medical in view of the patient's frailty and his good response to rate controlling drugs.

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